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Is Radiological Hand Osteoarthritis Associated with Atherosclerosis?: A Cross-Sectional Study in Turkish Women

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Osteoarthritis (OA) is the most common joint disorder that causes destruction of cartilage and decrease in joint space resulting in disruption of joint function. The hand is commonly involved in OA. Since there seems to be an etiologic association between hand OA and atherosclerosis, in this study, the degree of hand OA and atherosclerosis was analyzed in women.

A total of 61 women who are ≥ 50 years of age, independent of hand symptoms were enrolled in the study. Standard postero-anterior views of both hands were obtained using digital radiography. The radiographs were assessed by a single physician blinded for the patients. A total of 14 joints including the distal interphalangeal (DIP), proximal interphalangeal (PIP), metacarpophalangeal (MCP) and first carpometacarpal (1st CMC) joints of the thumb were assessed for radiographic osteoarthritis according to the Kellgren/Lawrence (K/L) score. An OA affected joint was defined as K/L score of ≥ 2 and hand osteoarthritis was defined as ≥ 3 joints of both hands affected with osteoarthritis. The severity of hand OA of each participant was indicated with the total K/L scores of all 14 joints of both hands. Gensini scoring was used to evaluate the patients for atherosclerosis severity. The mean Gensini scores of patients with hand OA was 21.00 ± 17.0 (median: 19) whereas in patients without hand OA was 10.95 ± 9.6 (median: 9) and the difference was statistically significant ($p=0.0045$). The degree of osteoarthritic joint involvement and Gensini scores showed a positive correlation ($r=0.31$). In conclusion, hand OA may be suggested as a finding that may be a red flag for a possible associated atherosclerosis. Patients with severe hand OA should be screened for atherosclerosis to prevent serious coronary artery disease related comorbidities.

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Prothrombin Gene Mutation (G20210A) is Not Associated with Nonvalvular Atrial Fibrillation with Ischemic Stroke in Turkish Population

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Background: Prothrombin G20210A is a genetic variant that approximately doubles or triples the risk of forming blood clots in the veins. The polymorphism is located in a noncoding region of the prothrombin gene (3' untranslated region nucleotide 20210), replacing guanine (G) with adenine (A). The variant causes elevated plasma prothrombin levels (hyperprothrombinemia). Prothrombin is the precursor to thrombin, which plays a key role in causing blood to clot (blood coagulation). Prothrombin G20210A can thus contribute to a state of hypercoagulability. Atrial fibrillation (AF) is the commonest sustained cardiac arrhythmia, which confers a high risk of mortality

and morbidity from stroke and thromboembolism. We aimed to investigate prothrombin G20210A mutation in patients with AF who have had a stroke than in healthy controls.

Methods: Prothrombin G20210A mutation was analysed in 70 patients with non-valvular AF who have had a stroke and 70 healthy individuals with no documented episode of AF matched for age, race and sex. Prothrombin G20210A mutation was identified by polymerase chain reaction (PCR) method. Distribution of prothrombin G20210A alleles (allele G, allele A) and genotypes (Normal (GG) genotype, heterozygous (GA) or homozygous (AA) mutant genotype) were determined in study population. Demographic characteristics and risk factors for AF and stroke were evaluated in the study groups.

Results: There was no significant difference with respect to age and gender between groups. There was no statistical difference in genotype distribution among the groups. The genotype distribution in nonvalvular AF who have had a stroke group was as follows: normal genotype (GG) frequency was 59 (84.3%) and heterozygous mutant genotype (GA) frequency was 11 (15.7%). The genotype distribution in control group was as follows: normal genotype (GG) frequency was 65 (92.9%) and heterozygous genotype (GA) frequency was 5 (7.1%). Homozygous genotype (AA) was not detected in both groups. There was no statistically significant difference among groups.

Conclusions: Our results suggest that prothrombin G20210A mutation appears not to be associated with nonvalvular AF with ischemic stroke in Turkish population.

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The Relation between MultiDrugResistance-1 C3435T Polymorphism and Pulmonary Hypertension in Patients with Chronic Obstructive Pulmonary Disease

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Objectives: Right ventricular (RV) dysfunction may develop in the course of chronic obstructive pulmonary disease (COPD) and it is important predictor of morbidity and mortality. The polymorphism of the gene of multidrug resistance-1 (MDR-1) responded to drug resistance have been associated with some worse clinical findings in patients with COPD. In this study, we aimed to investigate the relationship between MDR-1 C3435T gene polymorphism and RV dysfunction in COPD patients.

Methods: Forty one consecutive patients diagnosed priorly with COPD and hospitalized for acute exacerbation were enrolled. The polymorphism analysis was performed by Strip Assay technique. Right ventricular parameters were evaluated, and RV dysfunction was identified via transthoracic echocardiography. Patients were categorized into three groups according to gene polymorphism; MDR-1 CC (Wild Type, n=9), MDR-1 CT (Heterozygote mutant, n=21) and MDR-1 TT (Homozygote mutant, n=11).

Results: The study included 14 male and 27 female, mean age 65 ± 11 years. Mean systolic pulmonary artery pressure was 31.4 ± 8 mmHg in wild type group, 42.2 ± 12 mmHg in heterozygote mutant group and 46.5 ± 14 mmHg in homozygote mutant group ($p=0.027$) (Figure 1). Furthermore, presence of RV dilatation was significantly different among three groups (33%, 71%, and 100%, respectively, $p=0.005$). In multiple logistic regression analysis, MDR-1 C3435T gene polymorphism ($OR=9.000$, $p=0.019$) was independent predictor of RV dysfunction after adjustment of potential confounders.

Conclusion: The findings of this study demonstrate that MDR-1 C3435T gene polymorphism is associated with RV dysfunction in patients with COPD.

